

Imaging Findings of Eosinophilic Gastrointestinal Diseases in Adults

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ABSTRACT

Eosinophilic gastrointestinal (GI) disorders are a group of conditions marked by pathologic eosinophilic infiltration of one or multiple locations in the GI tract. Conditions include eosinophilic esophagitis, eosinophilic gastritis, eosinophilic enteritis, and eosinophilic colitis. The site and depth of eosinophilic infiltration of the GI tract usually determines clinical presentation. These conditions should be considered in the differential diagnosis for several GI symptoms, such as food impaction or dysphagia. Histopathology is the gold standard for diagnosis of eosinophilic disorders. Nevertheless, findings from endoscopy, barium studies, computed tomography or magnetic resonance imaging, can aid in the diagnosis, by allowing for earlier diagnosis as well as proper management. Eosinophilic gastrointestinal disorders are typically managed with corticosteroids or dietary elimination. A high index of suspicion is required for diagnosis as it can often be challenging.

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Introduction

Eosinophilic gastrointestinal disorders (EGIDs) are a group of disorders characterized pathologically by excess eosinophils in mural biopsies of one or several locations in the gastrointestinal (GI) tract,¹ in the absence of other causes for eosinophilia (such as drug reactions, parasitic infections, and malignancy).² Nomenclature and presentation of EGIDs depend on the site and extent of layer invasion of eosinophilic infiltration.³ EGID can be found at any age but is mostly seen in the third to fifth decades of life with a male predominance in adults and no gender predilection in children.² The gold standard for diagnosis is histopathology.⁴ Nevertheless, recognizing imaging characteristics of EGIDs can be helpful in achieving early diagnosis and appropriate management.

Eosinophilic Esophagitis

Eosinophilic esophagitis (EoE) is defined as an immune/allergy-mediated clinicopathologic condition, which is characterized by prominent eosinophilic infiltrate in the esophageal wall and symptoms of esophageal dysfunction.⁵ The reported prevalence of EoE is 34 cases per 100,000 children and 42.2 cases per 100,000 adults,⁶ and an overall prevalence of 78.8 per 100,000 individuals.⁷ There is

a male predilection for the disease in adults with a ratio of 3:1 (male to female).⁸ The majority of cases are detected during childhood. There is a peak prevalence between 30 and 44 years of age. In adults, the symptoms of EoE are mainly heartburn and dysphagia,⁹ although patients may also present with food impaction.¹⁰ The diagnosis of EoE depends on the clinical presentation and esophageal biopsies.

Diagnostic Criteria

Consensus recommendations based on a systematic review of the literature and expert opinion have led to the diagnostic criteria published by the American College of Gastroenterology. These criteria for EOE include: (1) presence of symptoms related to esophageal dysfunction (such as dysphagia, food impaction), (2) ≥ 15 eosinophils per high power field in the esophageal tissue, and (3) exclusion of other disorders associated with similar clinical, histologic, or endoscopic findings.^{11,12}

Pathologic Features

Pathologic characteristics of EoE can be divided into major and minor. Major features are not pathognomonic but considered characteristic and required for diagnosis, while minor features are non-specific but can assist in the diagnosis. Major features include eosinophilic micro-abscesses (4 or more eosinophils within the epithelium), increased intraepithelial eosinophils (more than 15 per high power field) (Fig 1), eosinophil aggregate in the surface layers

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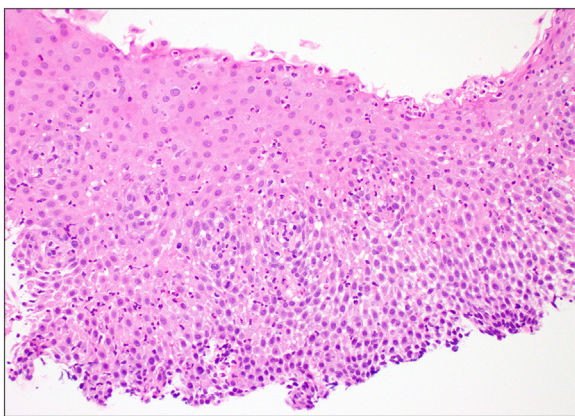


FIG 1. Histologic sections (hematoxylin-eosin stain) show esophageal squamous mucosa diffusely involved by eosinophils (greater than 100 per high-power field), with reactive features including basal cell hyperplasia and spongiosis (Color version of figure is available online.)

of the epithelium (“surface layering”), eosinophil degranulation, and surface sloughing of squamous cells and eosinophils. Minor features include lengthening of the lamina propria papilla, “marked” basal cell hyperplasia, increased intercellular edema, increased intraepithelial lymphocytes and mast cells, and increased lamina propria fibrosis and chronic inflammation.¹³

Endoscopic Findings

The EoE Endoscopic Reference Score (EREFS) is the classification system validated for EoE endoscopic findings, which is an acronym standing for the 5 major features: furrows (linear or longitudinal; 80%), edema, rings (64%), exudates and/or white plaques (16%) (Fig 2), and stricture (12%),^{3,14} and has shown a high degree of diagnostic accuracy.¹⁵

A normal appearing esophagus may also be a finding of EoE during endoscopic examination.^{16,17} Longitudinal furrows appear as long linear lines with subtle indentations parallel to the long axis of the esophagus.¹⁴ Esophageal exudates are whitish plaques that may represent eosinophilic inflammation and can be misinterpreted as candidiasis.¹⁴ Edema can occur in EoE and is characterized by loss of vascular pattern with generalized mucosal pallor.¹⁴ Fibrotic changes can be focal (≤ 1 cm in length), long (several centimeters in length), or a diffuse small caliber esophagus. Milder strictures with luminal diameter greater than 10 mm can be subtle endoscopically.¹⁸ Esophageal biopsy is still required to confirm suspected EoE.

Imaging Findings

Barium studies play a major role in the diagnosis of dysphagia and can be helpful to diagnosis a wide variety of causes including

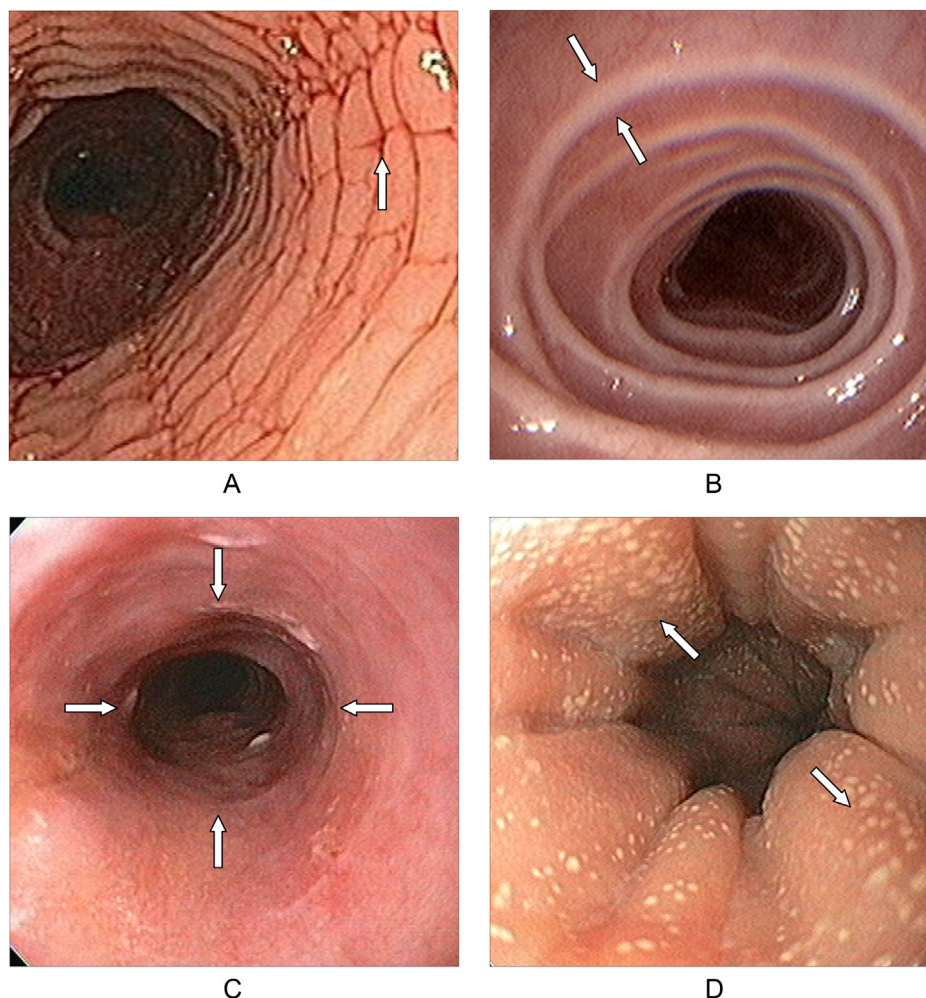


FIG 2. (A, B, C, D) Endoscopic images show linear or longitudinal furrows (arrow; A), concentric mucosal rings (arrows; B), small-caliber or narrow esophagus (arrows; C) and white plaques and/or exudates (arrows; D) (Color version of figure is available online.)

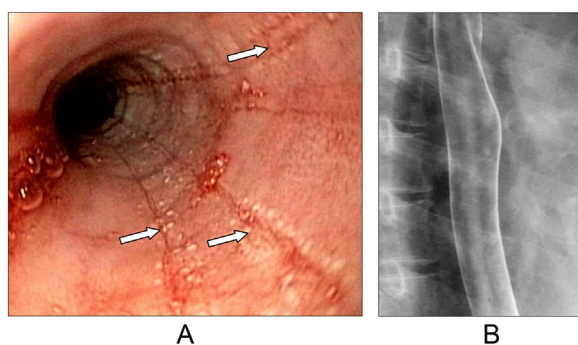


FIG 3. (A, B) Endoscopic image shows linear furrows (arrows; A) with normal esophagus on esophagram (B), illustrating a limitation of esophagram exams as furrowing is not identified (Color version of figure is available online.)

Schatzki's rings, achalasia, gastroesophageal reflux disease, strictures and cancers.¹⁹⁻²¹ Dysphagia is often a symptom in EoE and barium studies can be helpful to determine the underlying cause. In the setting of EoE, double contrast barium esophagrams can assist in the diagnosis, recognize subtle or diffuse strictures and evaluate changes that may be overlooked on endoscopy.²² Barium studies frequently show fixed rings and are more sensitive than endoscopy for the assessment of strictures and diffuse small-caliber esophagus.²³ Esophagrams are an important tool not only in identifying EoE, but in planning and following treatment, including assessing the need for dilation or stent placement.¹²

Endoscopic mucosal abnormalities in the setting of EoE such as furrowing, exudates and plaques are generally not well seen in barium studies even with optimal techniques (Fig 3).²⁴ However, other findings, such as the presence of esophageal rings which appear as distinctive concentric ring-like indentations should raise suspicion for eosinophilic esophagitis on barium studies (Fig 4).²⁵ These rings can occur as an isolated finding or may be associated with strictures or diffuse esophageal narrowing.^{21,26} The rings are thought to be a consequence of inflammatory scarring. The rings are typically over a short segment (<1 cm and more commonly between 2 and 5 mm), may pool barium contrast, horizontal in configuration, may extend partially or entirely across the esophagus, and often are multiple and sequential, creating a “stepladder” or “stacked coin appearance” (Fig 5), a finding also called “trachealization” of the esophagus.²⁶

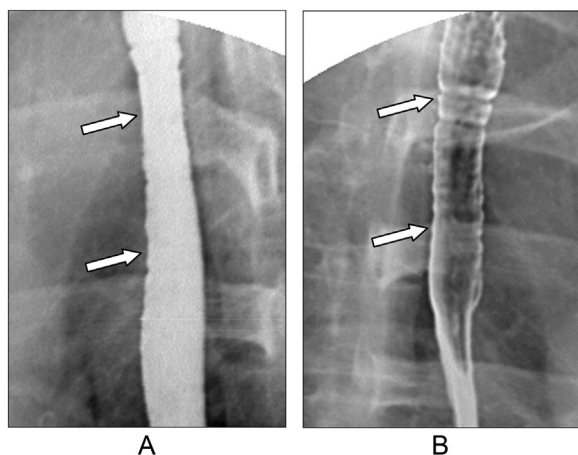


FIG 4. Thirty-year-old man with chronic sensation of food sticking in the upper esophagus. (A, B) Esophagram images show multiple concentric ring-like strictures and a narrow tubular esophagus compatible with eosinophilic esophagitis better seen on the double contrast images (arrows; B) compared to single contrast (arrows; A).

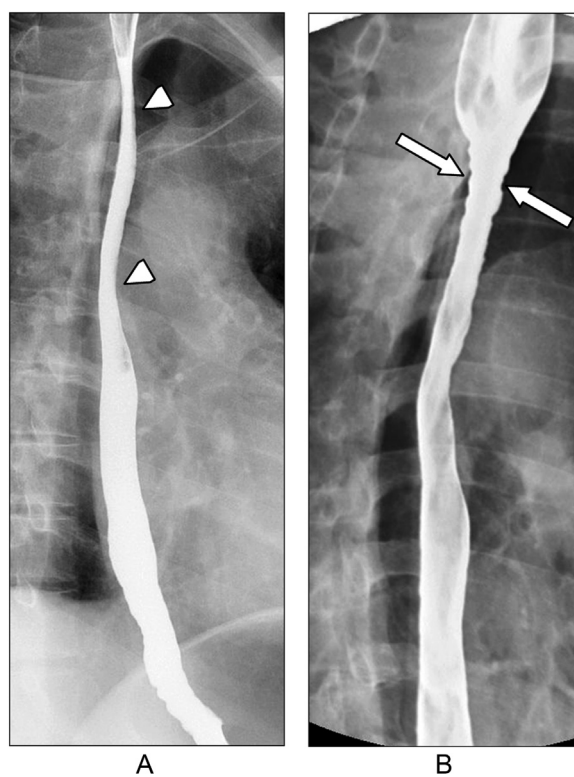


FIG 5. (A,B) Esophagram images show segmental strictures (arrowhead; A) in the upper-mid esophagus and “ringed” esophagus (arrows; A, B) with concentric, ring-like indentations of the esophagus like a “stack of coins,” compatible with eosinophilic esophagitis.

A high index of suspicion is required by radiologists as they may be the first physician to suggest the diagnosis.

Strictures typically manifest as concentric luminal narrowing with smooth contours and tapered margins (Figs 5 and 6).²¹ Esophageal remodeling with fibrosis and narrowing are common in EoE. Some cases of EoE may develop segmental strictures in the upper, mid, or less commonly, the distal esophagus. Esophageal strictures have been classified by the length of the narrowing as (1) rings (≤ 1 cm), (2) strictures (1-8 cm), and (3) small-caliber esophagus (> 8 cm). Luminal narrowing, rigidity of the wall, and ensuing stricture formation are suggestive of deeper layers of involvement.¹⁹ The presence of a stricture associated with eosinophilic infiltration is consistent with infiltration of the muscular layers of the esophagus.²⁰

A “small caliber” esophagus can suggest eosinophilic esophagitis (Fig 7).²¹ In the setting of a “short caliber” esophagus, a high index of suspicion is required as the diffuse narrowing of the esophagus can be difficult to appreciate on barium studies. A barium tablet can help detect subtle narrowing by getting stuck along its course to the stomach. Long-segment narrowing of the thoracic esophagus or small caliber esophagus are also more difficult to evaluate on endoscopy than a focal stricture, likely because of the subtle transitioning from unaffected to affected esophagus.^{22,23}

Esophageal intramural *pseudodiverticula* are an unusual finding and consist of a group of dilated excretory ducts of deep mucus glands in the esophagus. Esophageal intramural *pseudodiverticula* can be seen in EoE and are usually associated with increased intraluminal esophageal pressure proximal to a stricture.²⁷ These can also be associated with esophageal strictures from other causes,²⁸ gastroesophageal reflux disease (GERD), esophageal candidiasis, chronic esophagitis, and esophageal carcinoma.²⁹

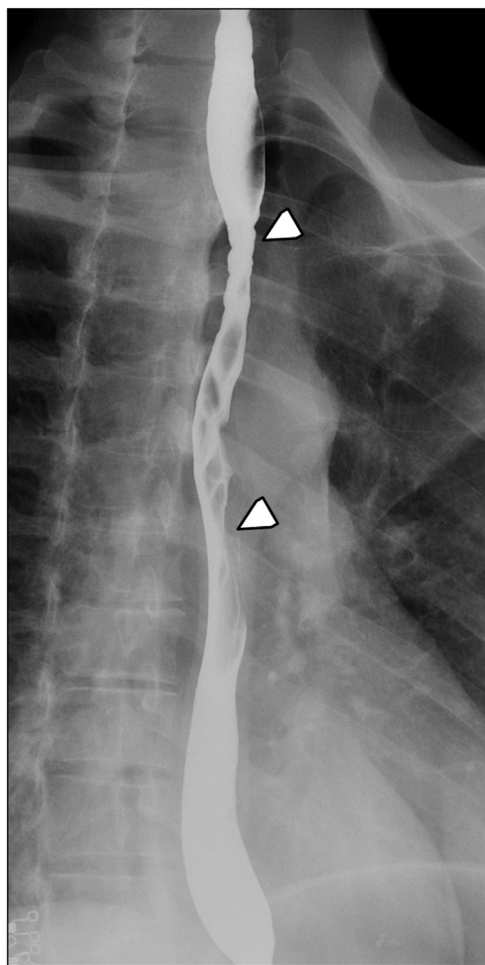


FIG 6. Fifty-two year-old woman with a history of dysphagia. Esophagram image shows significant esophageal narrowing and long stricture in the middle portion of the esophagus (arrowheads).

Pseudodiverticula are better appreciated with a single contrast esophagram,^{28,29} appearing as numerous, tiny (1-4 mm), flask-shaped outpouchings that may be diffusely distributed or clustered, and in profile appear to be “floating” next to the esophageal wall.²⁹

Computed tomography (CT) findings of EoE can be nonspecific, with diffuse esophageal wall thickening (Fig. 8) and sometimes associated findings of impacted food.^{4,30}

Complications

Esophageal perforation is a potentially lethal complication of EoE, that may develop as a complication of esophageal food bolus impaction, retching, or after mechanical dilation of esophageal strictures in EoE. Esophageal inflammatory changes and mucosal frailty, as well as remodeling, may increase the risk for spontaneous or iatrogenic esophageal perforation. Mucosal fragility of the esophagus is usually evidenced by tearing or shearing of the esophageal wall with passage of the endoscope. Fibro-stenotic changes in the esophagus lead to dysphagia and food impaction, which may lead to perforation. Esophagram with water-soluble contrast material followed by barium can be performed to detect esophageal perforation. CT findings of esophageal perforation include: extraluminal air, esophageal thickening (Fig 9), mediastinal, cervical, pleural, or pericardial fluid,

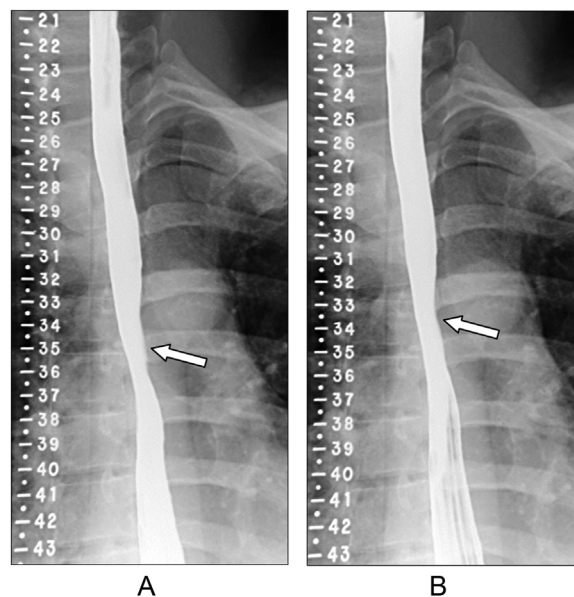


FIG 7. (A, B) Esophagram images show a small caliber esophagus. If table is not calibrated, a ruler may help to show stricture and for measurements. Also, a 12.5 mm barium tablet may be helpful to confirm narrowing/stricture.

though presence of fluid is less specific.³¹ Other potential complications of EoE include esophageal dysmotility, achalasia-like changes, intramucosal esophageal dissection, hepatic portal venous gas, and adrenal insufficiency.³²

Treatment

The aim of EoE treatment is to improve clinical symptoms, and prevent disease progression as well as complications³ by controlling contributing factors and the T helper 2 (Th2) lymphocyte response with the 3 “D’s” (“drugs,” “diet,” “dilation”) of therapy.^{33,34} The main “drug” treatment options include proton pump inhibitors (PPI), systemic or topical corticosteroids.³⁴ Dietary therapy is used to control environmental factors with an elemental formula diet (devoid of dietary protein) being the “gold standard” of the available options. Elimination diets directed by allergy testing and empiric elimination diets can be attempted as they have decreased cost, can be palatable, and avoid nasogastric or gastrostomy tubes.³⁵ Esophageal dilation treats the fibro-stenotic and structural changes and has been applied in adult EoE patients with strictures. The desired outcomes of treatment are to improve histologic eosinophilia to lower than the diagnostic threshold, aim for esophageal diameter of 16-17 mm, obtain endoscopic improvement, and improve symptoms like dysphagia.

Eosinophilic Involvement of the Gastrointestinal (GI) Tract

Eosinophilic involvement of the GI tract is part of the spectrum of diseases in which terminology and clinical presentation depend on the affected organ (location) and extent (mural layer of involvement) of eosinophilic infiltration. These include eosinophilic esophagitis, eosinophilic gastritis (Fig 10), and/or eosinophilic enteritis. Eosinophils may invade the mucosal layer, the muscular layer, or the subserosal layer of the GI tract, and symptoms differ according to the wall layer affected.³⁶ There is limited data on the prevalence of eosinophilic involvement of the GI tract, a recent review yielded prevalence of 6.3/100.000 for eosinophilic gastritis,

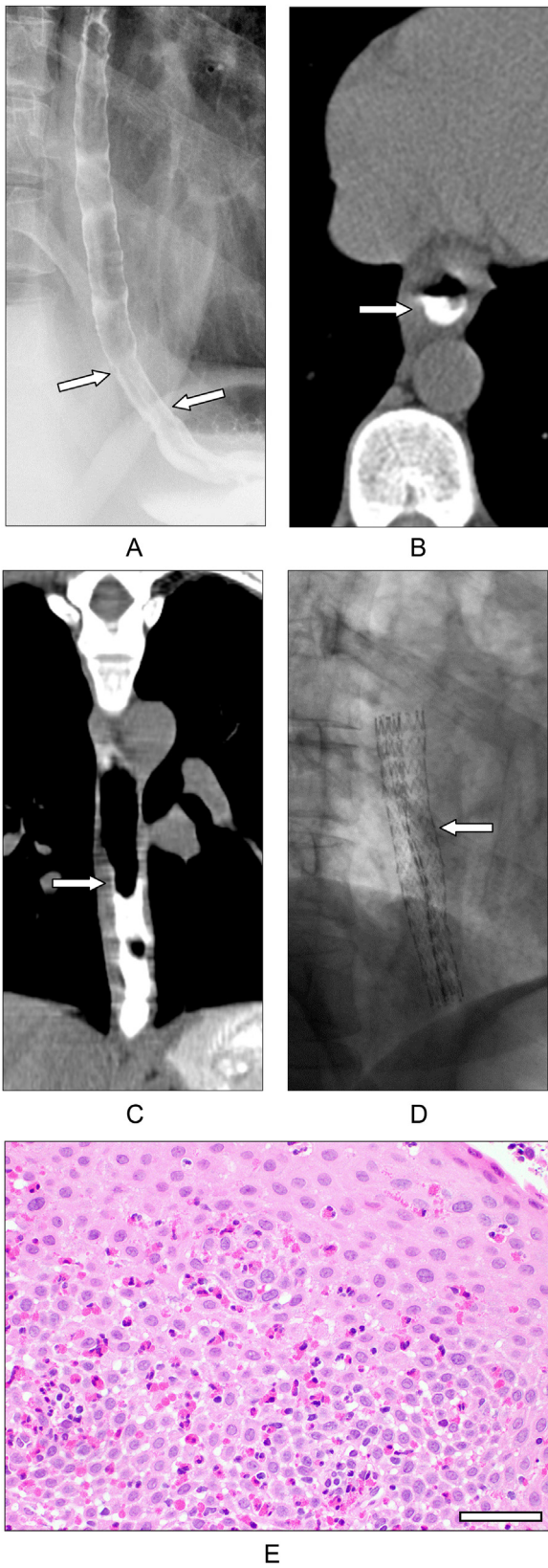


FIG 8. Thirty-eight year-old woman with a history of progressive dysphagia and EoE refractory to treatment. (A) Esophagram image shows a distal esophageal fixed stricture (arrows). (B, C) Axial (B) and coronal (C) CT with oral contrast images show moderate circumferential thickening of the distal esophagus (arrow) corresponding to area of stricture. (D) The patient ultimately required a stent across the stenosis (arrow). (E) Histologic section at high power (40x image- scale bar = 50 μ m; hematoxylin-eosin stain) shows esophageal squamous mucosa with spongiosis, diffusely involved by intraepithelial eosinophils (greater than 125 per high-power field-hpf) (Color version of figure is available online.)

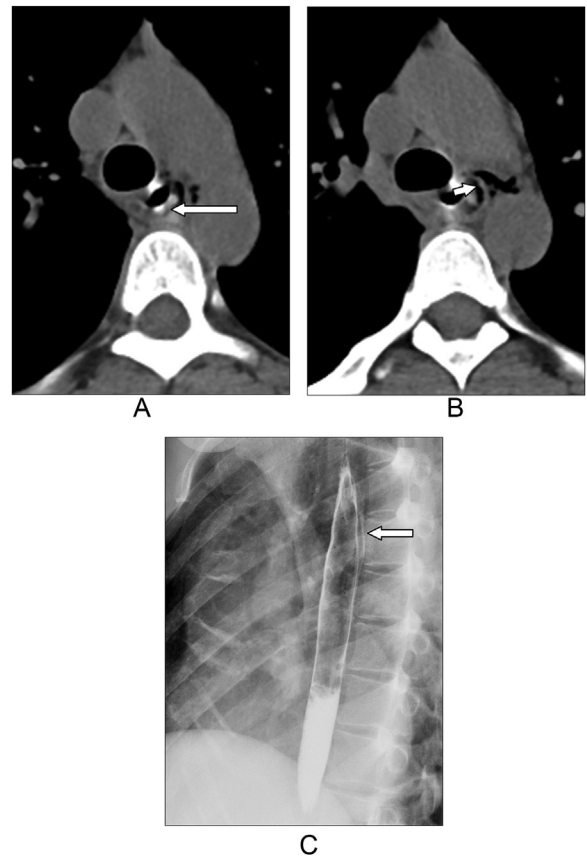


FIG 9. Thirty-nine year-old woman with a history of EoE and with recurrent dysphagia. (A,B) Axial CT with oral contrast images show outpouching of oral contrast in the mid left lateral esophageal wall (big arrow; A) and associated pneumomediastinum (small arrow; B), suggestive of focus of extravasation. Note the esophageal wall thickening. (C) Esophagram image shows contrast leak was identified within the esophageal wall in the thoracic esophagus (arrow), suggestive of an intramural esophageal dissection.

8.4/100,000 for eosinophilic enteritis, 3.3/100,000 for eosinophilic colitis in the USA. Estimates for EoE range from 4.5 to 10.4/10,000, thus non-EoE eosinophilic involvement of the GI tract are less common.³⁷

Primary eosinophilic gastritis, also referred to as idiopathic or allergic gastritis, may be isolated or associated with infiltration of eosinophils in other segments of the digestive tract, especially the esophagus, and can occur in the pediatric (most cases) or adult population.^{1,38} Eosinophilic enteritis is a rare disease

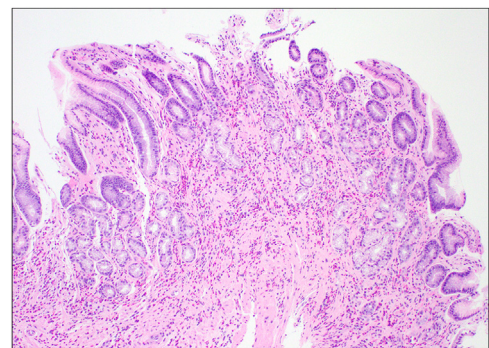


FIG 10. Low power histologic section (4x image- scale bar = 200 μ m; hematoxylin-eosin stain) shows gastric antral mucosa with extensive lamina propria involvement by eosinophils (Color version of figure is available online.)

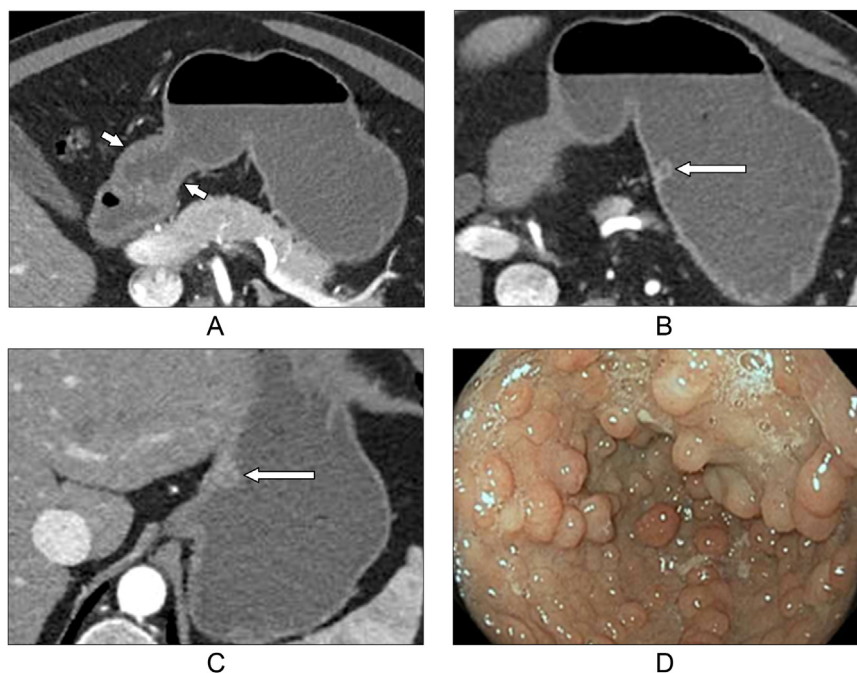


FIG 11. Forty- two year-old man with history of eosinophilic esophagitis and eosinophilic gastritis. (A) Axial contrast-enhanced CT image shows mucosal thickening and nodularity of the gastric antrum (small arrows). (B, C) Note the 2 benign-appearing inflammatory proximal gastric polyps (confirmed on biopsy) (larger arrows). (D) Endoscopic image shows eosinophilic gastritis with nodularity of the antrum from polyps in another patient (Color version of figure is available online.)

characterized by a pathologic infiltration of the intestine by eosinophils. Clinical presentation is mostly comprised of nonspecific gastrointestinal symptoms, and diagnosis requires pathological demonstration of eosinophilic infiltration of at least 1 intestinal segment, excluding other causes of intestinal eosinophilia.³⁹ The currently proposed histologic criteria for non-EoE eosinophilic involvement of the GI tract include >30 eosinophils in the stomach and/or duodenum.⁴⁰ Eosinophilic colitis is the rarest form of eosinophilic involvement of the GI tract, with most primary eosinophilic colitis cases being idiopathic and typically a diagnosis of exclusion. Pathogenesis is still unclear and this entity has poor response to treatment.⁴¹

Eosinophilic involvement of the GI tract appears to have a predilection for the distal antrum and proximal small bowel,^{42,43} though this may reflect easier endoscopic sampling access. Clinical manifestations vary according to the extent of involvement of different layers of the organ's wall. The most common form is the mucosal form, which is marked by abdominal pain, diarrhea,

malabsorption, protein-losing enteropathy, vomiting, blood loss in stools, and iron-deficiency anemia. The *muscularis* form is marked by eosinophilic invasion mostly in the muscle layer with thickening of the bowel wall, which may lead to gastrointestinal obstructive symptoms. The serosal form is the least common variant and is marked by exudative ascites with higher peripheral eosinophil counts.^{42,44,45}

Imaging exams such as barium studies, CT, or magnetic resonance imaging (MRI) of the GI tract may show thickening or nodularity in the antrum (Fig 11) and thickened and abnormal enhancing small³ or large bowel⁴⁶ (Figs 12–14). When the muscle layer is involved, imaging may show bowel strictures and decreased luminal diameter mostly in the distal antrum or proximal small bowel. These findings are not sensitive or specific for eosinophilic involvement of the GI tract and a normal imaging scan does not exclude this diagnosis.³ The mucosal form may also show, at imaging, polyps and ulcers. CT and MR imaging may also show ascites, obstruction, hypointensity or isointensity on T2-weighted image involving the bowel wall, restricted diffusion, and progressive enhancement.⁴⁷ Other features that may also be seen from bowel wall stratification include the “halo sign” (stratified bowel wall secondary to submucosal edema) which may assist in the differentiation of inflammatory and neoplastic lesions as well as excluding extra-intestinal disorders,^{4,48} but are nonspecific and can be seen in other inflammatory conditions. When the serosal layer is involved, serosal enhancement, ascites, omental thickening, and pleural effusion can be observed at imaging.^{4,30} In this serosal form, mesenteric lymphadenopathy with central necrosis may also be seen.^{30,48}

Treatment is based on PPI and *Helicobacter pylori* eradication (important but not primary), elemental diet, or targeted elimination diet, as well as drugs, such as corticoid, cromoglycate, montelukast, ketotifen,^{45,49} suplatost tosilate, mycophenolate mofetil (an inosine monophosphate dehydrogenase inhibitor).⁴⁵

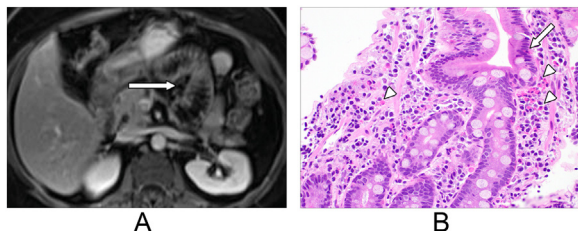


FIG 12. (A) Axial CT images show circumferential wall thickening in segments of the jejunum (arrow). (B) Histologic section with hematoxylin-eosin stain performed after CT scan shows small bowel with increased intraepithelial eosinophils (black arrowhead) in the lamina propria and rare intraepithelial eosinophil (white arrowhead) (Color version of figure is available online.)

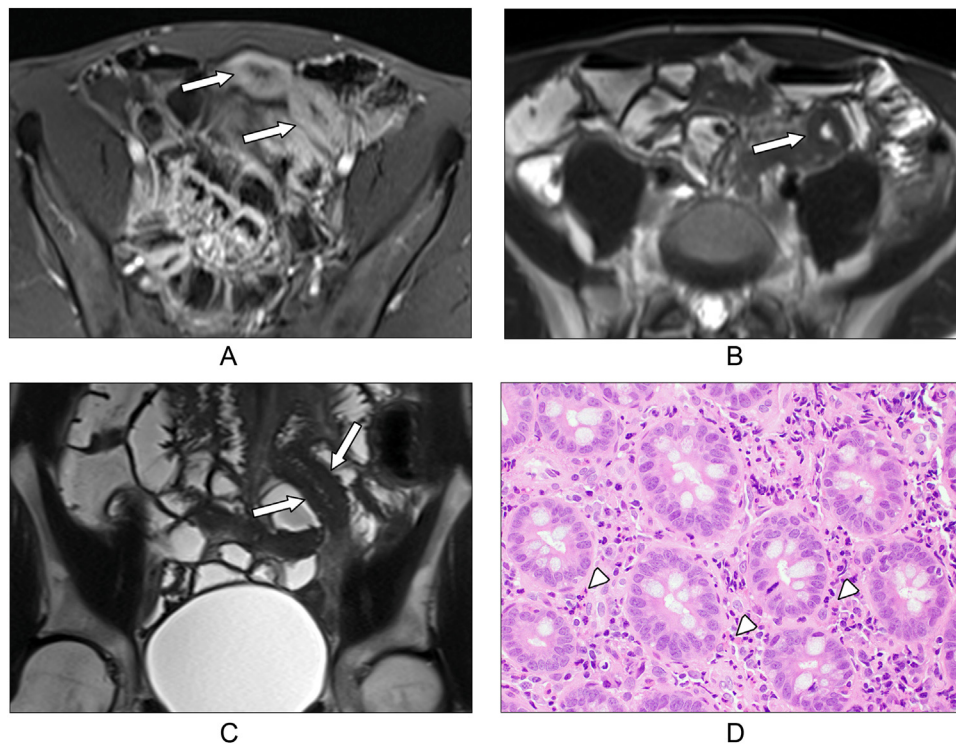


FIG 13. Twenty-five year-old man with history of abdominal discomfort, nausea, low weight. (A, B, C) MR enterography images show mid- small bowel demonstrating mild to moderate, circumferential wall thickening and enhancement on contrast-enhanced T1FS images (A) and thickening on axial (B) and coronal (C) T2-weighted images, compatible with eosinophilic enteritis. (D) High power (40x image- scale bar = 50 μ m; hematoxylin-eosin stain) reveals eosinophils in the lamina propria (arrowheads) in the areas of villous atrophy and superficial erosion (Color version of figure is available online.)

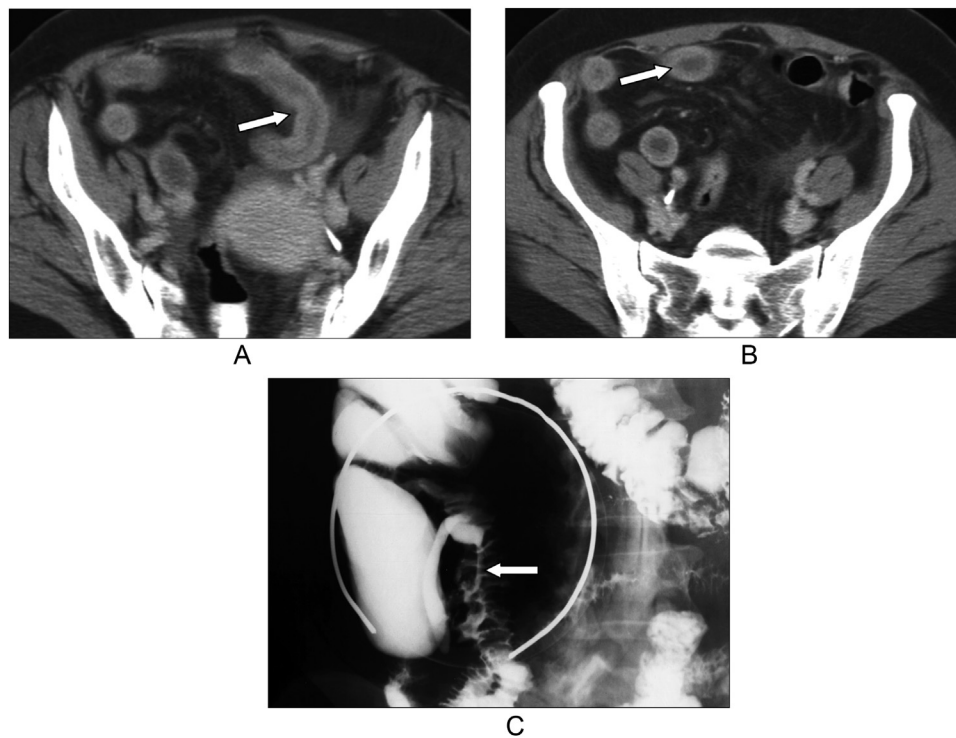


FIG 14. Eighty-three year-old woman with history of diarrhea, vomiting and diverticulosis. (A, B) Axial contrast-enhanced CT images show ascites, mild bowel wall thickening and mucosal enhancement of small bowel loops (arrow), also seen as nodular thickening of the terminal ileum on barium small bowel exam (arrow; C).

Conclusion

Eosinophilic gastrointestinal disorders are a group of inflammatory conditions marked by eosinophilic infiltration of one or multiple locations in the GI tract, simultaneously or sequentially. These disorders include eosinophilic esophagitis, gastritis, enteritis, and colitis. Imaging plays a key role in accurate identification of the affected site, thus allowing target biopsies to be effectively performed, as well as allowing proper management of these disorders to be implemented. Imaging also plays a major role in the identification of complications and in the follow-up examination of these patients. The mainstay of treatment is corticosteroids and dietary elimination.

Authors' Contributions

Authors are required to identify the contributions for which they are responsible.

1. guarantor of integrity of the entire study: Frank H Miller
2. study concepts and design: Camila L Vendrami, Frank H Miller
3. literature research Camila L Vendrami, Frank H. Miller, Linda Kelahan
4. clinical studies N/A
5. experimental studies / data analysis N/A
6. statistical analysis N/A
7. manuscript preparation Camila Lopes Vendrami, Linda Kelahan, Frank H Miller, David J Escobar, Ikuo Hirano
8. manuscript editing Camila Lopes Vendrami, Linda Kelahan, David J Escobar, Lori Goodhart, Nancy Hammond, Paul Nikolaidis, Guang-Yu Yang, Ikuo Hirano, Frank H. Miller

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